mental work accompanying intellectual occupations and posts involving heavy responsibility; (b) the emotional tensions that frequently accompany business, professional, and intellectual life; and (c) the inheritance of an ambitious or conscientious personality pattern. To sustain this argument it is necessary to show that these factors are much more potent in the affected classes. This has never been done; indeed, I believe (without scientific proof) that a dispassionate consideration of the matter exposes the concept as a fantasy of wishful thinking.

The reverse of the argument implies that the labouring classes in the wealthy countries and the great majority of the agrarian inhabitants of the Orient live some sort of idyllic existence, close to the soil, securely insulated from the fierce competitive, intellectual, and emotional burdens which grind the life out of those unfortunates whose lot it is to think, direct, and govern. It seems to me more likely that the stress factor operates in the reverse direction. The life of a physician in Birmingham is surely much less stressful than that of a peasant in the Yangtse Valley, with the ever-present menace of flood, famine, pestilence, and war; or of the tribesman in Equatorial Africa tortured by the taboo and sanction of primitive belief, to which are now added the tensions of organized political agitation. To subscribe at all to this factor one has to believe that the capacity to "feel" emotionally, to think deeply, to love, to hate, to fear, to hope, to aspire, and to despair are all much more highly developed in these materially favoured classes who get ischaemic heart disease. Basically, the intensity of all these intellectual and emotional activities, however much custom and education may alter their outward manifestations, is probably fairly uniform throughout the human family. Surely if these were fatal influences war, with such catastrophes as befell Norway, would greatly aggravate ischaemic heart disease. All available evidence is to the contrary.

The ready acceptance of this "stress-and-strain" concept is very understandable. It nourishes the *amour propre* of the believer and it is readily acceptable to the unfortunate victim and his relatives. It places ischaemic heart disease in the position of being the unjust reward of virtue. How much nicer it is when stricken with a coronary thrombosis to be told that it is all due to hard work, laudable ambition, and selfless devotion to duty than to be told it is due to gluttony and physical indolence. I know of nothing that conclusively disproves this psychological stress-and-strain concept; my contention is that so far there is little to support it, and much that makes it unlikely.

Conclusion

In conclusion I must emphasize that I have only briefly considered three of the many possible factors underlying ischaemic heart disease. There are many others, such as heredity and the important part played by essential hypertension with all its complex environmental background. It is important to recognize that such studies as I have considered merely indicate significant (in the statistical sense) correlations, which must not be taken as proving a causal connexion. They merely show how measurable environmental factors vary in relation to the incidence of ischaemic heart disease. It is easy to establish similar significant correlations with innumerable other factors whose causal relation is in the highest degree improbable. For instance, the progressive increase in ischaemic heart disease probably correlates closely with the rise in income tax, with the number of telephones in the community, or (inversely) with the decline in knowledge of the classics.

No other field of medical science illustrates so well the fundamental contribution of that academic discipline known as "social medicine," with its technique of the group inquiry, its searching examination of the validity of evidence, and its secure foundation on the mathematically disciplined philosophy of probability. It is right that the clinician should always keep before him the supreme importance of the individual patient, but he must never lose sight of

the importance of the patient as a unit in a complex pattern of biological adaptation. The application to diseases in general of the epidemiological techniques which have yielded such a rich harvest in the infectious diseases is the most exciting feature of current medical science.

The implications as regards prevention of such diseases as those we have considered, or of carcinoma of the lung, point to control lying in the hands of the individual rather than in those of the State. The complete control of water-borne infections has been achieved virtually without the knowledge and co-operation of the individual citizen, but anybody who has had experience of training soldiers to practise effective individual antimalarial measures will readily realize how hard it is to secure sustained effective co-operation. When such prophylaxis means avoiding attractive things and practising austere measures the propaganda problems become really formidable. However, the science of propaganda—as we know to our cost—also develops.

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COEXISTENT MYXOEDEMA HEART DISEASE AND PSYCHOSIS

BY

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The reports of the Committee of the Clinical Society of London (1888) and of Brun and Mott (1913) indicate clearly that an acute psychosis may arise spontaneously in myxoedema. More recent accounts (Stoll, 1932; Zondek and Wolfsohn, 1944; Asher, 1949; Miller, 1952) have focused attention on this fact. Indeed, Asher wrote, "Myxoedema is one of the most important, one of the least known, and one of the most frequently missed causes of organic psychoses." His report evoked editorial comment (*British Medical Journal*, 1949; *J. Amer. med. Ass.*, 1949; *Year Book of Endocrinology*, 1952) and has prompted us to describe two illustrative cases which we have treated in the past six months.

Our patients merited additional clinical and therapeutic interest, as one had associated "myxoedema heart" (Zondek, 1918), while the other had congestive cardiac failure with little cardiac enlargement. The combination of myxoedema heart disease and psychosis has not previously received special attention.

Case 1

A housewife aged 48, who had had previous good health, was admitted on September 15, 1953, with deeply cyanosed lips, orthopnoea, and anasarca. The main myxoedematous features were a creamy complexion with malar flush, dry skin, patchy alopecia of the scalp and outer eyebrows, dry

FIG. 1.—Teleradiogram of chest on admission showing classical myxoedema heart.

brittle hair, a deep toneless voice, and slight deafness. The secondary sex characters were normal. She was mentally dull but rational.

The remaining salient physical signs were mainly confined to the cardiovascular system, and included venous congestion, and sacral and ankle oedema. The apex beat could not be located, but on percussion the area of cardiac dullness was increased to the left and right. The cardiac sounds 1 and 2 were muffled but present at all areas and were accompanied by a short apical systolic murmur. The pulse was regular at a rate of 62 a minute, of good volume and without pulsus paradoxus. The radial vessel wall did not feel thickened and there was no locomotor brachialis. The blood pressure was 150/80. Other systems were normal apart from basal crepitations in the chest and a swollen abdomen with a small ascitic effusion and a firm liver edge palpable 3 cm. below the costal margin in the mid-clavicular line.

The immediate teleradiogram (Fig. 1) revealed an enormous cardiac silhouette with sharply stencilled borders and shadowing at the lung bases. The remaining investigations are set out in the Table, where the initial results are compared with those obtained one week before discharge from hospital, when they had reverted towards normal. Neither cardioscopy nor pericardial paracentesis was performed. The urinary neutral 17-ketosteroid excretion was 1.4 mg. a day. The blood Wassermann reaction was negative.

Immediately after the initial tests, treatment was begun with thyroid extract, $\frac{1}{2}$ gr. (32 mg.) daily. After three weeks on this dose, acute psychotic signs first appeared, while the cardiac silhouette was virtually unchanged. The lung bases were clear and she could now lie flat comfortably. Her mental state warranted urgent certification. She was wildly confused, aggressive, and abusive, with delusions of persecution. Suicidal attempts rapidly followed. They included

Results of Investigations During First Week Compared With Those Obtained Three Months Later

	Early	Late
Thyroid extract daily dose ..	Nil	8 gr. (0.52 g.) for 2 months
Weight	12 st. 3 lb. (77.6 kg.)	9 st. 4 lb. (59 kg.)
Haemoglobin	79%	82%
B.M.R. (mean value)	-19%	+6%
Serum cholesterol	204 mg. per 100 ml.	152 mg. per 100 ml.
E.C.G.: Rate	60/min.	75/min.
QRS voltage	Very low	Normal
Axis deviation	Nil	Left
T waves	Flat or inverted in all leads	Upright. Low voltage in some leads

swallowing smelling-salts, a hair-grip, and buttons torn from her nightdress. She attempted suffocation under her pillows and ruthless self-mutilation by trying to cut her wrists and by violent scratching of her forearms. Next she chewed up the corner of a bed-sheet and tried to swallow it. Further suicidal attempts seemed imminent. Instead of immediate certification we resorted to more vigorous thyroid therapy, realizing that her cardiac state was a relative contra-indication, but predicting a dramatic resultant improvement of her psychosis (Asher, 1949; Miller, 1952). Single daily doses of 1, 2, and 4 gr. (0.06, 0.13, and 0.26 g.) respectively were given for the next three days, aided by persuasion and sedation, without producing cardiovascular distress. Her mania persisted and next day the dose was increased to 8 gr. (0.52 g.) daily. Two days later she was remarkably placid and rational. Further convalescence was uneventful. She continued to take 8 gr. (0.52 g.) of thyroid extract daily without evidence of overdose. Her mental state was normal after one week on this higher dose. She now jokingly referred to her recent bizarre behaviour.

On December 10 she was mentally and physically fit and was discharged home on 8 gr. (0.52 g.) of thyroid extract daily, and keen to resume her household and canteen duties. Her periods had now returned after 10 months' absence. The pulse rate was 80 a minute, the blood pressure 160/95, and the cardiac silhouette was almost within normal limits. The apex beat was easily located in the fifth left interspace just beyond the mid-clavicular line. The cardiac impulse was thrusting in type and the cardiac sounds were pure and of normal intensity. The abdominal signs were unaltered.

FIG. 2.—Teleradiogram of chest three months after thyroid extract 8 gr. (0.52 g.) daily.

She has since attended the out-patient clinic weekly. On January 16, 1954, her daily dose of thyroid extract was reduced to 6 gr. (0.4 g.) as she complained of palpitations, flushes, excessive perspiration, and insomnia, but not of anginal pain. The pulse rate was 110 a minute, the blood pressure 210/120, and the cardiac silhouette (Fig. 2) remained slightly larger than normal. A fortnight later these symptoms recurred and the daily dose of thyroid extract was further reduced to 4 gr. (0.26 g.), at which level it remained until the time of writing (March, 1954). The pulse remained steady at 70 a minute and the blood pressure at 150/90. Her weight was 9 st. 11 lb. (62.1 kg.); she was symptom-free and able to carry out her duties efficiently. Although slight cardiac enlargement persisted radiologically, there had been no further signs of congestive cardiac failure. She received no digitalis throughout. If the photographic test for myxoedema (Asher, 1949) had been applied before and after a few weeks on thyroid therapy the differences would have been impressive.

Case 2

A housewife aged 58 was admitted to hospital on October 31, 1953, after alternating phases of semicoma and maniacal outbursts during the previous day. She had signs of early congestive cardiac failure and complained of diarrhoea of three years' duration, which was later shown to be the spurious type due to faecal impaction. She was slightly built and diminutive (4 ft. 6 in. (135 cm.) in height and 10 st. 2 lb. (64.4 kg.) in weight) with a microcephalic appearance and a retroussé nose. The myxoedematous features closely resembled those in Case 1. Further examination revealed minimal orthopnoea and venous congestion, moderate ankle and slight sacral oedema, bradycardia (60 a minute) with thickened radial vessel wall and hypotension (115/75), but no other abnormal cardiovascular signs. Apart from the respiratory system, where there were basal crepitations, other systems were normal.

She had never received thyroid medication, and her previous history (from her relatives) revealed that for 17 years she had been subject to manic depressive episodes, two of which had led to prompt mental certification. The various episodes included apathy, mental inaccessibility, fugues, a paranoid state, and assault on nurses. During a previous admission to hospital (July, 1953) for congestive cardiac failure, this disappeared with rest in bed and mersalyl injections but without digitalis medication. During the determination of the basal metabolic rate, however, she became acutely maniacal and suspicious of this procedure, necessitating her third certification before thyroid medication was begun.

On the present admission the possibility of "pituitary myxoedema" was considered in view of her lapses into semicoma, hypotension, and her spare build. This point was never settled, because of her uncooperative state. The determination of urinary excretion of neutral 17-ketosteroids, although of no great differential diagnostic value, had to be deferred because of her frequent urinary incontinence. There was, however, no other supportive evidence of decreased activity of the anterior pituitary gland, as her secondary sex characters were normal, she had a family of five whose ages ranged from 16 to 30, and there was no history of post-partum haemorrhage. She had undoubted arteriosclerosis, but there was no history of a previous coronary thrombosis to explain the hypotension.

The serum cholesterol was 400 mg. per 100 ml. A chest x-ray film showed that the cardiac silhouette was not appreciably enlarged. The electrocardiogram showed low voltage with iso-electric or inverted T waves in all leads. The determination of the basal metabolic rate was deferred to avoid a repetition of mania, while it was also appreciated that any result obtained could hardly have been regarded as "basal" so long as there was residual cardiac failure or the possibility of agitation. The Wassermann reaction was negative in the blood, and a similar result had previously been obtained on the cerebrospinal fluid.

Treatment was begun cautiously with $\frac{1}{2}$ gr. (32 mg.) of thyroid extract daily, and this dose was gradually increased to 3 gr. (0.2 g.) over the next six weeks. We refrained from giving a larger dose in view of the mild psychotic picture, the arteriosclerosis, the congestive cardiac failure, and finally the remote possibility that her myxoedema was of pituitary origin. These latter patients may react unfavourably to even small doses of thyroid extract with adrenal cortical failure (Escamilla, 1950). Digitalis therapy was withheld, as it was not strongly indicated in view of the bradycardia and the mild congestive cardiac failure.

After three weeks' thyroid medication the congestive cardiac failure had entirely disappeared. There was striking improvement in her mental state. This was not expected in view of the long history of psychosis. There were no further attacks of semicoma. Her conversation was now moderately intelligent. She was less suspicious and introverted and more docile, while for the first time in 10 years she read the newspaper. The pulse rate had increased (70 to 80 a minute), but the hypotension remained (110/70). Her weight had fallen to 8 st. 2 lb. (51.7 kg.) and the serum cholesterol level to 124 mg. per 100 ml. The basal metabolic rate had now a mean value of plus 10%. The cardiac size radiologically, and the electrocardiogram, were completely normal. She was discharged on January 21, 1954, feeling fit and free from spurious diarrhoea and urinary incontinence. She has since attended the medical out-patient clinic fortnightly. One month after discharge from hospital her daily dose of thyroid extract was reduced to 2 gr. (0.13 g.), as she was complaining of occasional flushes, but there was no other evidence of thyroid overdosage. She is now able to carry out most of her household duties and shopping. Her attitude to other patients in the ward and to its nursing staff has altered, as she now visits the ward regularly in a friendly fashion after her attendance at the clinic.

Discussion

Our two cases illustrate the dramatic effect of thyroid therapy on both myxoedematous psychosis and myxoedema heart disease. In treating the latter, it is customary to be cautious with thyroid medication (McGavack *et al.*, 1945), using an initial daily dose of about $\frac{1}{2}$ to $\frac{3}{4}$ gr. (8 to 16 mg.) of the extract, as these patients are especially apt during treatment to lapse into acute congestive cardiac failure or to develop acute coronary insufficiency if larger doses are used. This caution was later waived to some extent in the treatment of our first case when acute psychosis predominated.

It has been shown (Lange, 1944) that capillary permeability is increased in myxoedema. This would explain best the occurrence of a complicating pericardial effusion, which Gordon (1929) first reported. This effusion, however, may not always be a constant, early, and major factor in myxoedema heart (Bell and Beck, 1953), despite the suggestion of Kern *et al.* (1949), for it is also recognized (La Due, 1943) that an additional cause for this massive cardiac silhouette is a dilatation of all the cardiac chambers.

Although myxoedema heart with congestive cardiac failure has been reported, this association is rare (Blumgart and Freedberg, 1952). In our first patient, who was considered to have myxoedema with a pericardial effusion and congestive cardiac failure, the possibility that the anasarca was at least partly due to hepatic cirrhosis and partly to congestive cardiac failure was entertained but not explored. The disappearance, during thyroid therapy, of the oedema but not of the abdominal swelling with its persistent hepatic enlargement and signs of minimal ascites suggests that both aetiological factors were operative. Pure myxoedematous ascites is reabsorbed during thyroid therapy (Paddock, 1950; Tracey and Donovan, 1951; Ferayorni and Sprague, 1952), while the same result occurs in anasarca due to congestive cardiac failure in myxoedema (Fahr, 1928) in which digitalis rarely restores circulatory efficiency (Andrus, 1953). It is also recognized (Hurxthal, 1935; McGavack *et al.*, 1945; Means,

1948) that, as in Case 1, following thyroid therapy in myxoedema heart, the cardiac silhouette may not return to within normal limits for several months.

Concerning myxoedematous psychosis, it is interesting that this appeared spontaneously and for the first time in Case 1 after three weeks on $\frac{1}{2}$ gr. (32 mg.) of thyroid extract daily, thus suggesting the necessity of a larger dose for its control. There have been frequent pleas (Stoll, 1932; Zondek and Wolfsohn, 1944; Asher, 1949; Miller, 1952) for a wider recognition of myxoedema as a cause of acute insanity, especially since failure to recognize it may lead to denial of the one treatment which can be both specific and dramatic. Schwimmer *et al.* (1947), in their review of the clinical manifestations of 40 cases of myxoedema, stressed the fact that the diagnosis is often missed primarily because it is not entertained, while a further important contributory factor is an all too frequent reliance on a complete clinical picture, whereas many of the unmistakable classical signs may be absent. A further drawback, as pointed out by Bartels (1950), is that profound myxoedema may occur in the presence of only a slightly lowered basal metabolic rate or a normal plasma cholesterol value. The latter was demonstrable in our first patient.

There is no constant psychotic picture in myxoedema. Most of these patients have been women. The personality changes cover the wide range of mania, irritable and suspicious character, self-accusation, delusions, hallucinations, melancholia, and a schizoid state, but rarely, as in Case 1, suicidal attempts. At other times there may be lapses into semicoma (Committee of the Clinical Society of London, 1888; Stoll, 1932), as in Case 2. In this transient hibernation it is conceivable, if evidence of vascular disease were found or the myxoedematous signs were less striking, that the cause could be readily misdiagnosed or missed. This cerebral state should not readily be identified with the pre-terminal coma, described by Summers (1953), which no treatment so far has reversed. Concerning the cause of acute myxoedematous psychosis, there has been little study but some speculation.

Mott (1915), however, examined the histological appearances of the brain tissue obtained from the necropsy of such patients. He observed that perinuclear chromatolysis in the cerebral cortex was a constant feature of the few cases studied, but this finding is non-specific. It has also been described in such toxic states as lead encephalitis and alcoholic psychosis, and this led him to suppose that the myxoedematous psychosis was mainly "due to a lack of functional energy which may be correlated with the marked disappearance of the Nissl substance (kinetoplasm), possibly associated with a toxic condition." This would imply that the reduced hormonal influence of the thyroid gland might result in the accumulation of a metabolite which was toxic to the brain. No such substance has so far been identified, and his alternative suggestion of an absence of some substance essential for nerve-cell metabolism merits equal consideration.

Zondek and Wolfsohn (1944), however, attributed the improved mental status after thyroid therapy to "dehydration." They had previously demonstrated that, during thyroid therapy in myxoedema, a diuresis with the mobilization of extracellular fluid results. More recently, the cerebral circulation and metabolism in myxoedema have been investigated by Scheinberg *et al.* (1950), who found that the cerebral blood flow and oxygen and glucose consumptions were all decreased in patients with myxoedema, compared with normal values. Thyroid treatment restored these lowered levels towards normal. These workers suggested that the mental changes in myxoedema were the result of decreased cerebral oxygen and glucose metabolism. They also confirmed the electroencephalographic findings of Bertrand *et al.* (1938) in myxoedema. These include typical low voltage, a lack of alpha waves, and an absence of reaction to a light stimulus, but only one or more of these were regularly present in any one patient. Even, however, if alpha waves are present there is a good correlation of the

alpha rate with the metabolic rate in myxoedema (Ross and Schwab, 1939). This test may have occasional value in the confirmation of diagnosis.

Myxoedema may evade diagnosis, may masquerade as other medical or mental disorders, or may fail to be confirmed if reliance is restricted to one test. If myxoedema heart or myxoedematous psychosis or both are present, failure to recognize these or a misdiagnosis means failure to supply the only treatment which is beneficial and at times even dramatic, while without treatment the prognosis is serious. It was believed that in Case 1 a psychiatric disaster was averted and the patient has become a useful citizen, while it is hoped that future certification will be spared in Case 2.

Summary

Two cases of coexistent myxoedematous psychosis and heart disease are described. Both features in both cases responded well to thyroid extract therapy. A brief review and discussion of these two important complications of myxoedema are given. The difficulties in the clinical diagnosis and confirmation of diagnosis are discussed, and the theories on causation of myxoedematous psychosis are briefly mentioned.

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The Anglican Diocese of Nyasaland maintains a number of medical stations situated round the southern half of Lake Nyasa, in Portuguese East Africa, as well as in Nyasaland itself. There are seven small hospitals, two small leprosy settlements, and four other clinics and dispensaries. An annual total of nearly 2,000 in-patients and 35,000 out-patients are dealt with. In 1953 there were over 235,000 out-patient attendances at the medical stations. Most of the hospitals, including that on Likoma Island, are administered by nursing sisters. The headquarters of the medical officer to the Anglican Diocese in Nyasaland is at Malindi. The present medical officer is retiring soon, and the Universities' Mission to Central Africa is endeavouring to find a successor.